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Case report

A case of hypocalcemia-related epilepsia partialis continua

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ABSTRACT

Epilepsia partialis continua (EPC) is a syndrome clinically defined as continuous spontaneous jerking confined to one part of the body, sometimes aggravated by action or sensory stimuli, occurring over hours, days or even years. In adults the more frequent recognized cause of EPC is an acute cerebrovascular disease. Acute severe hypocalcemia is a highly epileptogenic ionic disturbance, abnormally increasing neuronal excitability. In this short communication we describe the first probable case of acute hypocalcemia-related EPC. Eight months after a left parietal lobe cardioembolic stroke, a 74-year-old woman experienced a generalized tonic-clonic seizure for the first time in her life, at the beginning of a Clostridium difficile enterocolitis. Four days later, while the abdominal symptoms were clinically improving, continuous semi-rhythmic jerks of right face, shoulder and arm began suddenly. Despite several appropriated antiepileptic treatments those involuntary movements did not cease. On routine biochemical examination we noted a total calcium serum level of 1.2 mmol/L (normal range 2.1–2.8 mmol/L), not previously known. After intravenous calcium gluconate supplementation, the jerks started to fade, disappearing completely as a total calcium serum level of 1.9 mmol/L was reached. Two separated CT brain scans did not reveal new cerebral lesions. Neurophysiological studies did not show any cortical activity related to jerks. Taken together, the treatment refractoriness and the clinical improvement after ionic imbalance correction point towards a highly possible role of hypocalcemia in sustaining the activity of a previously silent epileptogenic focus.

1. Introduction

Epilepsia partialis continua (EPC) is a syndrome that is clinically defined by continuous spontaneous jerking confined to one part of the body, sometimes aggravated by action or sensory stimuli, occurring over hours, days or even years. The pathophysiology and anatomic basis of EPC are only partially understood. Evidence suggests a cortical origin in the majority of cases, even though in some patients a subcortical origin cannot be excluded. Cortical reflex myoclonus appears to be the main mechanism underlying EPC.[1,2] The most frequently encountered cause of EPC in adults is an acute cerebrovascular disease.[1,3] Acute severe hypocalcemia is a highly epileptogenic ionic disturbance, because it can enhance neuronal excitability. In this short report we are presenting a probable case of acute hypocalcemia-related EPC.

2. Case report

Eight months after the development of right emiparesis and dysarthria occurred secondary to left parietal cardioembolic stroke, a 74-year-old woman came to our hospital's Emergency Department accusing acute abdominal pain and diarrhea. A few hours later she suffered two generalized tonic-clonic seizures for the first time in her life. Once admitted to the Emergency Care Unit, she was given an antiepileptic therapy with Levetiracetam 1000 mg/day. During the following days the patient suffered from abundant dysenteric discharges and an antibiotic treatment with oral Vancomycin was needed, due to the positivity of Clostridium difficile toxins in the fecal specimens. After four days, while the abdominal symptoms were clinically improving, she was suddenly affected by continuous semi-rhythmic jerks of right face, shoulder and arm. The involuntary movements occurred at an almost constant 1 Hz frequency; they were not stimulus sensitive and diminished in amplitude during sleep. The patient always remained lucid and cooperative, though she was unable to voluntarily move the affected limb. A 10 mg Diazepam bolus produced a transitory effect over jerks, but causing a respiratory arrest, so no other attempts were made. After that a 1.5 mg/kg infusion of Valproic Acid was started, with no evident clinical benefit, even after Phenytoin 300 mg/day was added. Two brain

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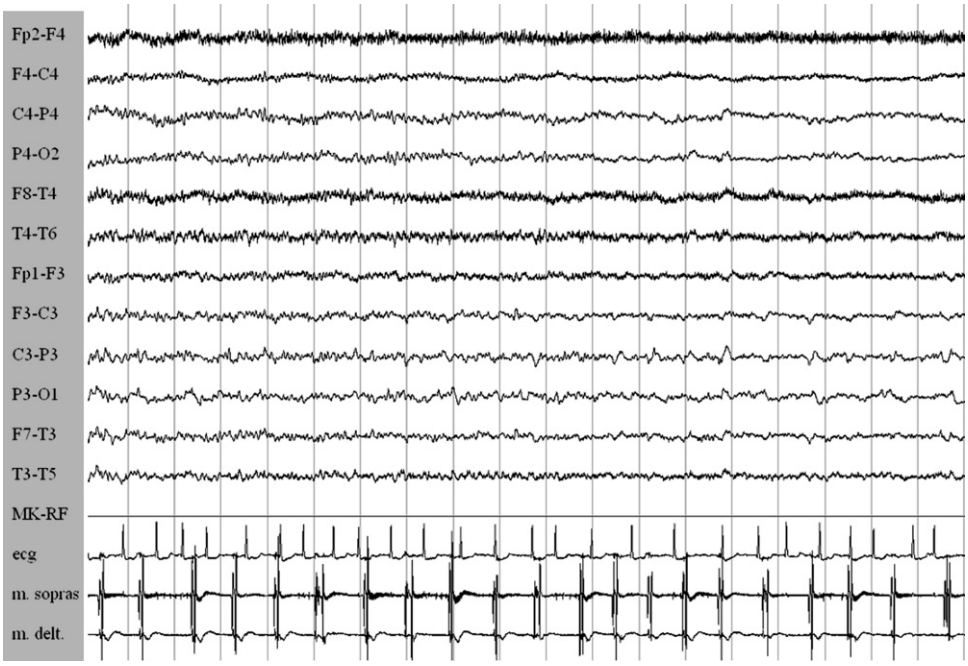

Fig. 1. Short title: EEG poligraphy. Detailed legend: continuous quasi-rhythmic twitching of right deltoid and supraspinatus muscles. EEG reveals only an irregular theta activity over the left parietal region, without clear epileptiform correlates.

computed tomography (CT) scans, performed at a distance of 48 h from one an other, revealed only the long-standing ischemic lesion. Three days later the patient was admitted to our Neurological Unit. On a routine biochemical examination we noted a total calcium serum level of 1.2 mmol/L (normal range 2.1–2.8 mmol/L), not previously reported. Shortly thereafter we introduced a calcium gluconate intravenous supplementation. As total calcium serum levels raised to 1.7 mmol/L, the spontaneous activity became progressively less frequent, losing its rhythmic quality. The jerking ceased completely, as her calcium level reached 1.9 mmol/L. After we managed control over those involuntary movements, the patient underwent wide investigations to find a possible explanation for her calcium ionic imbalance. Severe hypocalcemia was likely to have been provoked by a moderate senile vitamin D deficiency, together with inadequate food intake and malabsorption due to the acute Clostridium intestinal infection. The patient was discharged, maintaining only Levetiracetam 1500 mg tid antiepileptic therapy. No further seizures occurred so far, though a mild worsening of her right emiparesis is evident, as compared to pre-hospitalization clinical status.

3. Discussion

In many cases EPC has a cortical origin, however it cannot be excluded that subcortical structures play a role. Myoclonus can be diagnosed and classified into cortical, brainstem, spinal, and unclassified.[4] On a clinical base this appears to be an example of spontaneous myoclonus. The EEG trace doesn't show clear epileptiform correlates, as shown in Fig. 1. However, the absence of EEG spikes in association with myoclonus does not entirely exclude the possibility of a cortical origin. Thus we proceeded to the jerk-locked back averaging technique, since it might disclose a cortical spike not detectable by surface EEG. Back averaging failed to detect a spike preceding motor activity, but this doesn't necessarily bring to a subcortical origin of the myoclonus; one possible explanation for back averaging failure is EEG signal's attenuation by the skull. Next we decided to perform somatosensory evoked potential (SEP) since most patients with cortical myoclonus show extremely enhanced cortical waves, but they didn't reveal abnormal features. Giant potentials are highly related to stimulus-sensitive myoclonus, and less constantly with spontaneous ones. Therefore, even the absence of giant SEPs cannot completely rule out a cortical origin in our patient and the myoclonus remains unclassified.[1,4,5]

We propose a causal link between EPC and hypocalcemia for several reasons. First of all, from a biochemical standpoint there is enough convincingly in vitro evidence that extracellular low Calcium solutions enhance neuronal excitability: sodium voltage-gated channels open at lower values of membrane potential and after-hyperpolarization currents are diminished, while the depolarizing persistent Sodium current is facilitated.[6,7] Second, from a clinical point of view, hypocalcemia seemed to sustain the jerking activity. Indeed no antiepileptic drug had effect on myoclonus, with the partial exception of Diazepam, while a surprisingly rapid decline in clinical manifestations occurred as Calcium blood levels became progressively close to physiologic values. Taken together, treatment refractoriness and clinical improvement after ionic imbalance correction point towards a probable role for hypocalcemia in sustaining the activity of a previously silent epileptogenic focus.

Conflict of interest

None declared.

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